

Gastritis

The highlight of the World Congress in the area of gastritis was the introduction of a 'new' classification of gastritis by a working party chaired, somewhat surprisingly, by JJ Misiewicz from the Central Middlesex Hospital in London, United Kingdom, and consisting of a well chosen and primarily European panel. The aim was "to provide a simple, easy to apply, comprehensive and comprehensible classification that is flexible, correlates with pre-existing ideas, and can be sensitive to treatment effects." Although showing signs of perhaps being relatively hastily put together, with discrepancies between the published document and the data presented, and therefore showing signs of being relatively untried, the system is actually quite logical and proves very similar to that which many have been using for some time. It tries to establish what is going on and why, and emphasizes that gastritis has etiological, endoscopic and histological components, the final diagnosis being a compendium of all three. Distribution remains critical, but alphabet soup (A, B, AB, C) is dropped in favour of 'pangastritis' or 'antral' or 'corporal' inflammation.

Briefly, the history establishes that the patient is taking nonsteroidal anti-inflammatory drugs or has Crohn's disease, for example. Endoscopy is primarily descriptive, perhaps regrettably called 'endoscopic gastritis' despite the fact that many things that are red are not inflamed while many things that are inflamed histologically are normal endoscopically. Histology adds information regarding the presence and grading of chronic and/or acute inflammation, the distribution coming from the two recommended biopsies from antrum and corpus. The final diagnosis is given as the etiology where known, the topography and the morphology, in that order, producing diagnoses such as '*Helicobacter pylori*-associated antral moderate acute and severe chronic inflammation' – a bit of a mouthful; nevertheless it is all there. A variety of

special forms of gastritis (eosinophilic, granulomatous, etc) are handled similarly when possible, eg, 'bile-associated reactive gastritis of the antrum' (although this is not usually histologically inflamed).

Histological grading is purely arbitrary and subjective, which means that each pathologist will develop his or her own criteria. This will lead to some degree of confusion in patients in whom re-endoscopy and biopsy are carried out to assess therapy, unless the same pathologist sees all of the biopsies or ensures that pre- and post treatment biopsies are compared. Four illustrations – eg, a certain amount of inflammation or less is normal; more is abnormal and mildly inflamed; a certain amount of inflammation or more is at least moderate; and a certain amount or more is severe – might have been a useful guide for both acute and chronic inflammation.

A point of confusion is that in the published report all inflammation is graded on the dominant cell type as acute or chronic. This seems to have been dropped at the presentation, and rightly so, because in *H pylori*-associated gastritis, at which this classification is primarily aimed, there is invariably more chronic than acute inflammation so that the early decrease in acute inflammation following treatment, as well as the pre-treatment biopsy, would all be called chronic.

No justification is given for the recommended two biopsies from antrum and corpus other than the occasional mild focality of *H pylori*-associated disease, but how this disparity is actually handled in practice was unclear. The decision to omit a biopsy from the angulus deliberately, because such biopsies are more difficult to interpret, guarantees that the question of multifocal chronic atrophic gastritis, said by some to be an important precursor of gastric cancer in high risk areas, remains unresolved, without stopping those interested from adding this biopsy.

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Despite the ease of pointing out the potential flaws of the Sydney system, to quote Ashley Price who gave the histological viewpoint: "It is easy to knock religion, but just try inventing a new one." The working party has made a sound start. This classification has as its major virtue the combination of all aspects for a final diagnosis, which is clearly the only way to go, and should catalyze a clinicopathological correlation. The classification is primarily an information-gathering tool; hopefully at the next Congress in Los Angeles in 1994 many of the problems arising will have been identified and smoothed out.

Although space does not permit detailed coverage, a sec-

ond working party chaired by Guido Tytgat from Amsterdam, The Netherlands gave a superb symposium on "*Helicobacter pylori*: Causal agent in peptic ulcer disease", which dealt with all aspects of pathogenesis and treatment. Even the relatively brief working party report is about the best recent summary encountered and is highly recommended.

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